The brain was edematous and weighed 1,320 gm. There was a cavity (Figure 3) in the inferior surface of the right temporal lobe, and the pia arachnoid was thickened in the region of the pons. The ventricles were not enlarged and there were no anomalies of the circle of Willis. There was no evidence of hemorrhage and no purulent material was present.

In microscopic study of sections of the lung it was noted that several bronchi had foci of lymphocytes at the periphery. There was an increase in connective tissue in several places and granulomata were present. In some of the latter there was caseation necrosis in the center. Giant cells of Langhans type were also found in the granulomata. Spherules were seen in specially stained preparations.

A few granulomata with Langhans giant cells were found in the fibromuscular stroma of the prostate, and spherules were also seen in the gland in specially stained preparations. The pia arachnoid of the cervical cord was infiltrated with lymphocytes and several granulomata were present. A spherule with endospores was seen in the connective tissue near a Langhans giant cell. There was congestion of the intrinsic blood vessels and dilation of the perivascular spaces of the cerebellum and similar findings of granulomata in the pia arachnoid. In the right temporal lobe of the brain there was a large area of liquefaction necrosis. Granulomata were observed in the pia arachnoid. There were also spherules with endospores and Langhans giant cells. Granulomata were noted in the choroid plexus of the midbrain and there was widening of the perivascular spaces and congestion of the intrinsic vessels.

Coccidioides immitis did not grow on cultures of material from the brain.

DISCUSSION

As far as could be ascertained there are in the literature only two cases in which coccidioidal pulmonary cavitation and coccidioidal meningitis were coexistent. Jenkins and Postlewaite[1] reported one case and mentioned another reported by Smith, Beard and Saito.[2] There seemed to be nothing in the present case that would help explain why pulmonary cavitation and dissemination of coccidioidomycosis rarely are concomitant.

SUMMARY

A fatal case of coccidioidal meningitis with coexistent coccidioidal pulmonary cavitation is presented. Positive reaction to coccidioidin skin tests, positive complement-fixation on coccidioidal serological tests and microscopic findings of spherules with endospores in the pia arachnoid substantiated the diagnosis. Negative results of viral serological examinations of Manitoux skin tests and cultures for acid-fast bacilli helped to rule out tuberculosis and the viral encephalitides.

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Congenital Hepatomegaly

Report of a Case

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IN MOST of the reported cases of congenital anomalies of the liver the abnormality is in the number of lobes; rarely, cases of accessory livers are reported.

Congenital anomalies are related to a disturbance of the normal embryological growth. Normally the primordial outgrowth destined to become the secretory and duct system of the liver, together with the gallbladder, arises ventrally from the entodermal lining of the gut and is known as the hepatic diverticulum. As the hepatic tubules grow in size and number, they extend between the two layers of the splanchnic mesoderm, which constitutes the ventral mesentery at this level. As growth continues, the two layers are separated over the surface of the liver and give rise to the fibrous connective tissue capsule of the liver, as well as to the interstitial connective tissue of the liver lobules and smooth muscle layers of the duct system.

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One of the congenital anomalies of the liver, Riedel's lobe, was described by Riedel[6] in 1888 as a "tongue-shaped projection descending from the anterior surface of the anterior margin of the right lobe of the liver to below the umbilicus." Unlike appendicular lobes of other types, Riedel's lobe is always associated with gallbladder enlargement.

Accessory livers have been described in the pleural cavity,[5] in the greater omentum,[4] in suspensory ligament[3] and in an amniotic hernia at birth.[1]

Another anomaly of the liver, heteroptosis, was described by Eppinger[2] as a phenomenon similar to a "floating" spleen or kidney. In this condition the degree of ptosis may vary widely. In almost all cases of heteroptosis, relaxation of the anterior abdominal muscles, with some degree of diastasis of the recti muscles, is present. Herniation of the liver mass through the anterior abdominal wall has been observed in some cases, and displacement of other abdominal organs may occur. In the majority of cases, the liver is freely movable and can easily be returned into its normal position beneath the dia-

phragm, while the mass tends to slide down again on deep inspiration.

Most anomalies of the liver do not cause symptoms, but are observed at autopsy. In cases in which symptoms do occur, they may vary from acute abdominal signs to the presence of a nontender, freely movable mass in the abdomen. In the case here reported they were of the latter order.

REPORT OF A CASE

A 2-year-old boy was observed by one of the authors because of poor eating habits. On examination a mass was felt in the left upper quadrant of the abdomen. The mother had not noticed this mass.

The patient was of normal growth and development for his age. The skin was pale, and it was noted that the skin became mottled when the child stood for a short time. The chest was of the funnel type, with pronounced flaring of the diaphragm. The abdomen was protuberant. Some diastasis of the recti muscles was present. The mass in the left upper quadrant of the abdomen was sharply defined and freely movable. It extended from approximately 2 cm. to the right of the midline at the right costal margin to a point approximately 4 cm. above the left iliac crest. Upon application of slight pressure in the left costovertebral area, the outline of the point of the mass pressing against the anterior abdominal wall could be seen.

Erythrocytes numbered 4,120,000 per cu. mm. of blood and the hemoglobin content was 12.0 gm. per 100 cc. Leukocytes numbered 11,400 per cu. mm. Cell fragility was within normal limits. The platelet count was 271,000 per cu. mm. The serum protein content was 6.2 gm. per 100 cc., and the albumin globulin ratio was 4.2:2. No abnormality was noted in biopsy of a specimen of bone marrow. Results of urinalysis were within normal limits. Upon pyelographic examination a bifurcate kidney and double ureter on the left were observed, and a configuration interpreted as an "unsuccessful" attempt at doubling on the right. Renal function appeared to be good. A gastrointestinal study with barium showed an essentially normal upper gastrointestinal tract and the presence of a large mass covering almost half of the left lumbar area. No esophageal varices were seen. No displacement or pressure on other organs was noted in the x-ray studies. The absence of the hepatic shadow on the right side of the abdomen was commented upon.

Exploratory laparotomy was done and the left lobe of the liver was found to extend across the entire abdomen from the midline to 5 cm. below the left costal margin. The right lobe was extremely small and extended for only 2 cm. to the right of the midline. The ligamentous attachments were in normal position. The gallbladder was also in its normal place, but was enlarged. From the left margin of the left lobe, a tongue-like projection was found, extending downward as far as the left iliac crest. Biopsy of specimen of the liver was done and no abnormality was noted. No other abnormalities were found at the time of exploration of the peritoneal cavity. The patient did well postoperatively and was discharged from the hospital.

SUMMARY

The case presented is one of congenital hepatomegaly in which the left lobe of the liver was enlarged and had an appendicular lobe attached to it. The conditions were similar to those described by Riedel,[6] except that the hepatic anomalies were on the left instead of on the right side of the abdomen. In addition, a bifurcate kidney and double ureter were present on the left side.

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Paraffinoma of the Penis

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INJECTIONS OF PARAFFIN were first done on organs of the male urogenital tract. Gersuny[3] began the practice in 1899 by injecting paraffin into the scrotum to replace testicles removed because of tuberculosis. Successful results were reported. Later, paraffin injections were used to advantage in the treatment of urinary incontinence; the vesical mucous membrane was infiltrated with paraffin. The procedure was adopted widely and was extended to include injections for hernias, atrophic rhinitis, cleft palate, permanent separation of divided nerves, and to improve or alter facial deformities.

Heidingsfeld[4] in 1906 first described tumors resulting from paraffin injections. Quérnu and Pérol[5] summarized six cases of paraffinoma of the penis from the foreign literature. In five of the six cases the tumors were caused by vaseline injections given to enlarge the penis. All five patients lost erective power, probably because of the inelastic sclerotic shell of fibrous tissue which developed. In 1949, a case of paraffinoma of the penis was reported by Bradley and Ehrgott.[1] This was believed to be the

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